Complicated Burn Resuscitation

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KEYWORDS

• Heart failure • Cirrhosis • Inhalation injury • Acute renal failure

KEY POINTS

- New technology and new information are helping improve the care of thermally injured patients during resuscitation.
- Reliance on old criteria and old-school judgment to guide resuscitation still has a role in management.
- There is an absolute combination of injury severity and lack of physiologic reserve that is uniformly fatal.

INTRODUCTION

More than 4 decades after the creation of the Brooke and Parkland formulas, burn practitioners still passionately argue about which formula is the best. So it is no surprise that there is a lack of consensus about the conundrum of trying to resuscitate a thermally injured patient with a significant comorbidity such as heart failure or cirrhosis or how to resuscitate a patient after an electrical or inhalation injury or a patient whose resuscitation is complicated by renal failure. To a large degree, all of these scenarios share a common theme in that the standard rule book does not apply. None of these patients can be resuscitated in a usual or standard fashion. All will require highly individualized resuscitations. There will be scenarios where advanced monitoring may be in the patient's best interest, and there are cases where careful titration of therapy to the old-school clinical signs of cardiac output—mental status, urine output and skin perfusion—may be ideal.

HEART FAILURE

A thermally injured patient with significant pre-existing heart failure is truly an acute on chronic failure problem. The baseline pathophysiology of the patient's cardiac disease is added to the hypovolemia and myocardial depression of an acute burn. Heart failure

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is estimated to affect 2 to 3 million Americans, with a rate that significantly increases after the age of 45 and by the age of 65 affects men and women at equal rates.[1] The etiologies of heart failure are primarily ischemic, but also include mechanical dysfunction such as valvular disease and the nonischemic cardiomyopathies from viral, alcoholic, metabolic, and idiopathic causes. The clinical hallmarks of the failing myocardium are dysrhythmias, systolic dysfunction, and elevated diastolic tone. These manifestations of heart failure are driven by dysregulated calcium oscillations in the myocardium, resulting in high diastolic intracellular calcium and a remodeled heart where the myocardial architecture does not have the appropriate myocyte alignment secondary to the loss of the normal interstitial fibrillar collagen network. The failing heart provokes a neurohumoral response, such that resting sympathetic tone and baseline norepinephrine levels are elevated, and the renin-angiotensin-aldosterone axis is activated. Predictors of poor survival with heart failure are left ventricular (LV) ejection fraction less than 30%, LV end diastolic diameter greater than 7 cm, LV end diastolic volume (LVED) greater than 130 mL, serum norepinephrine greater than 600 pg/mL, narrow pulse pressure (systolic blood pressure, diastolic blood pressure [SBP-DBP/SBP <25%]) and New York Heart Association class 4 symptoms.

Burn injury unleashes a massive inflammatory cascade of histamine, prostaglandins, thromboxane, serotonin, catecholamines, and cytokines. These agents have the effect of reducing cardiac output and increasing systemic and pulmonary vascular resistance that is not corrected by normovolemia.[2–4] At baseline, the failing heart is living in an environment of increased sympathetic tone and stimulated neuroendocrine state, so an acute burn adds further to this stress and may identify some patients who have no further cardiac reserve. One of the strongest prognostic factors for survival in heart failure is whether patients respond to dobutamine. Patients who are maximally endogenously stimulated and who do not have any remaining functional reserve do not increase their cardiac output under dobutamine stimulation. These patients have a decreased rate of survival.[5] The stress of resuscitation may unmask these unfortunate patients early into their burn resuscitation.

Invasive monitoring of the heart by use of a Swan Ganz catheter, serial echocardiogram (ECHO) or other means is necessary for these brittle and often difficult-to-assess patients. The use of pulmonary artery (PA) catheters is declining in intensive care units due to concerns of insertion-related complications, potential infection risk, and data that suggest that the information obtained is not always used effectively. Initial enthusiasm for goal-directed therapy utilizing Swan-Ganz catheters to obtain ideal oxygen delivery and oxygen consumption endpoints has been dampened by the increased use of fluids needed for this strategy and the phenomenon of fluid creep seen in many burn centers in the last 15 years.[6] A survey of burn centers in Europe, the United States, Canada, and Australia showed that less than 8% of burn units use PA catheters frequently.[7] Unfortunately, the uncommon use of these catheters means that when the burn practitioner really needs these data (and in the heart failure patient undergoing acute burn resuscitation these data are critical), the team is less facile with obtaining and interpreting the data from the Swan-Ganz catheter. A new disposable minitransesophageal echocardiography device is now available that can be left in place for up to 72 hours. Initial use of this device has shown both safety and efficacy, but the costs are significant.[8,9] Serum B-type natriuretic peptide, a protein secreted from a stretched myocardium, has been shown to correlate with volume overload, and because it can be measured in a serial manner, it may be considered a way to screen for this state during resuscitation.[10] Careful volume resuscitation to restore adequate LVEDV and to keep right atrium pressure less than 8 and pulmonary artery